Original Article

Left Atrial Volume Index to Left Ventricular Ejection Fraction Ratio Predicted Major Adverse Cardiovascular Event in ST-Elevated Myocardial Infarction Patients during 8 Years of Follow-up

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Abstract

Objective: It is crucial to determine the high-risk group in ST-elevated myocardial infarction (STEMI). Left ventricle ejection fraction (LVEF) and left atrial volume index (LAVI) are the well-established parameters for risk prediction. However, major adverse cardiovascular events (MACEs) may be predicted less than actual when LVEF or LAVI are in the normal range. It was investigated LAVI to LVEF ratio (LAVI/LVEFr) for more accurate MACE prediction. **Methods:** Patients with STEMI were included in the study. LAVI and LVEF were obtained at admission. The LAVI/LVEFr was calculated as LAVI dividing by LVEF. The composite primary endpoint of the study was all-cause mortality and new-onset heart failure for 8 years follow-up. **Results:** A total of 176 patients were divided into two groups according to the presence of MACE. MACE (+) group consisted of 70 (39.7%) patients who were older and more likely to be male. While LVEF ($P < 0.001$) was lower, LAVI ($P < 0.001$) and LAVI/LVEFr ($P < 0.001$) were higher in MACE (+) group. Age ($P = 0.003$), serum creatinine ($P < 0.001$), and LAVI/LVEFr ($P < 0.001$) were independent predictors of MACE. **Conclusion:** Combined usage of LAVI and LVEF (LAVI/LVEFr), increased age, and serum creatinine level were the independent predictors of MACE during 8 years of follow-up in STEMI patients.

Keywords: Left atrial volume index, left atrial volume index to left ventricle ejection fraction ratio, left ventricle ejection fraction, ST-elevated myocardial infarction

Introduction

Despite modern medical and interventional treatment options and early revascularization by the primary percutaneous coronary intervention (p-PCI), ST-elevated myocardial infarction (STEMI) is still responsible for a large number of deaths globally.[1] Therefore, determining the high-risk patient groups is important to provide earlier and intensive treatment options. Despite many clinical factors that have been identified to predict clinical outcomes, these scoring tools consisted of many clinical factors, results were controversial, and were studied for <5 years.[2] Since clinical conditions such as myocardial fibrosis, ventricular remodeling, recurrent MI, ventricular dilatation, and arrhythmias occur in a long period, a longer follow-up course could bring more reliable prognostic value for major adverse cardiovascular events (MACEs) prediction.

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Transthoracic echocardiography is routinely used for both risk stratification and outcome estimation in STEMI patients. Left ventricular ejection fraction (LVEF) is one of the major determinants of cardiovascular adverse events during short- and long-terms follow-up.[3] On the other hand, the impaired atrial function has emerged as a focus of cardiovascular research in recent years. Left atrial volume index (LAVI) was shown to be a substantive marker of left atrial (LA) function in previous studies. Moreover, increased

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LAVI was found to be a strong predictor of morbidity and mortality in acute myocardial infarction (MI).[4,5] However, in patients with preserved ejection fraction or atrial size, the poor outcome might be underestimated than it is.

Proportional clinical predictors such as neutrophil/lymphocyte and C-reactive protein/albumin ratios were shown to have better predictive value compared to their single usage in outcome estimation.[6,7] Hence, it was aimed to examine the role of LAVI to LVEF ratio (LAVI/LVEFr), a novel echocardiographic marker, in MACE prediction in STEMI patients who underwent p-PCI, during the long-term follow-up.

Methods

Study population

This is a prospective and observational study. A total of 176 consecutive STEMI patients who admitted to the hospital between March 2010 and May 2012 were included in the study. Informed consent forms were obtained from all patients. The study protocol was in line with the Helsinki declaration and approved by the local ethics committee.

STEMI diagnosis

The STEMI diagnosis was created according to the current guidelines. Patients with typical anginal symptoms and an elevation of the J point at least 0.2 mV in two consecutive V1, V2, or V3 leads or 0.1 mV in other leads were considered as STEMI. In addition, ST depression in lead V1 through V3 was considered as posterior MI.[1] The diagnosis was confirmed by diagnostic coronary angiography.

Demographical and laboratory data

The clinical characteristics including detailed medical history and physical examination were obtained from each patient by experienced cardiologists at admission. All the data were stored in the database of our institution.

Hypertension (HT) was defined by considering the following parameters: (i) patients who were diagnosed with HT by the international diagnostic code and/or (ii) patients who were taking one or more of the antihypertensive medications for at least 6 months. Diabetes mellitus (DM) was diagnosed according to at least one of the following criteria: i) History of DM and taking any anti-diabetic medication, (ii) randomly measured blood glucose value of 200 mg/dL or higher, (iii) blood glucose level of 126 mg/dL or above after at least 8 h of fasting, and (iv) A1c value of 6.5% or higher.[8] The presence of hyperlipidemia (HL) was defined according to age- and sex-adjusted percentiles from the National Health and Nutrition Examination Survey III data.[9] The body mass index was calculated according to the weight/height (cm) ^{2} formula.

All patients were recruited to routine outpatient clinics on the 1st, 3rd, 6th, and every year at regular intervals. Physical examination, echocardiographic findings, and laboratory data of each patient were recorded at these examinations.

Transthoracic echocardiographic evaluation

Two-dimensional M-mode transthoracic echocardiography was performed for all patients by the EPIQ 7C ultrasound system (Philips, Best, the Netherlands) before coronary intervention. LVEF was calculated according to the modified Simpson's method. LA volume was measured at end-systolic apical 2- and 4-chamber frames. Planimetric trace was conducted to measure the LA border within the LA wall and mitral annulus borderline. Pulmonary veins' ostium and LA appendage were not included in the measurement. LAVI was calculated by the following formula: LA volume/body surface area. LAVI/LVEFr was calculated by dividing LAVI by LVEF.

Coronary angiography and percutaneous coronary intervention

Coronary angiography was conducted immediately after hospitalization using the Judkins technique in all patients. Left anterior descending and circumflex coronary arteries were viewed from at least four different angles and the right coronary artery from at least two different angles. The images were transferred to the digital media for the quantitative analysis. The coronary artery with total occlusion was revascularized with the coronary balloon and/or stent immediately after imaging. Thrombolysis in myocardial infarction (TIMI) flow and collateral vessel status were evaluated and recorded at the database system of the hospital. Intervention to the total occluded coronary artery at first angiogram was determined as the revascularization strategy. All obtained data, coronary angiography views, and results were recorded in the database of ours institute.

All patients were treated medically according to the current guidelines. Patients were given the loading dose of clopidogrel and acetylsalicylic acid before the procedure. At the beginning of the procedure, 5000 or 10.000 IU intravenous heparin was administered according to the weight of the patients.[10] Coronary stenting directly or followed by balloon angioplasty was performed where eligible. After the procedure, patients were followed in the intensive coronary unit until stabilization is achieved.

Exclusion criteria

Pulmonary embolism, any type of malignancy and history of radiotherapy or chemotherapy, cardiac surgery with any indication, congenital heart disease, endocrine disorders, collagenous vascular disease, acute or chronic renal failure, end-stage liver disease, active inflammatory disease, history of cerebrovascular disease, moderate-to-severe valvular heart disease, myocarditis, and cardiomyopathy were determined as the exclusion criteria.

In addition, patients whose MACE-related data could not be acquired were excluded from the study. Written consent forms for PCI were obtained from all patients, and those who refused PCI or transthoracic echocardiography were not included in the study.

Clinical follow-up

The composite primary outcome of the study was all-cause mortality and new-onset decompensated heart failure (HF) for 2 years' follow-up period. Mortality and HF data were obtained by the query of the hospital and national databases and direct phone calls or face-to-face interviews with patients or relatives of relevant patients. All patients were examined at the 1st, 3rd, 6th, and every year regularly. Typical HF symptoms including shortness of breath, swelling of ankles, palpitation, weakness, and jugular venous fullness, pulmonary congestion, and peripheral edema were assessed at these examinations. Patients with the above-mentioned symptoms and physical examination findings and with LVEF are under 40% were accepted as HF.

Statistical analysis

SPSS software package (version 23.0, SPSS, Inc., Chicago, IL, USA) was employed to analyze the obtained data. P <0.05 was considered to have statistical significance. A 5% type-I error level was used to infer statistical significance. The normality assumption of data was assessed by the visual (histograms, probability plots) and analytical methods (Kolmogorov–Smirnov/Shapiro–Wilk's test). Levene's test was used to check the homogeneity of variances. While the mean ± standard deviation scheme was used to represent the continuous variables, the percentages were used to present the categorical variables. The Chi-square or Fisher's exact test was conducted with the purpose of comparing the categorical groups. While the two-tailed Student's *t*-test was used for normally distributed parameters, the Mann-Whitney U-test was performed for the nonnormally distributed continuous variables. The univariate regression analysis was carried out to assess the effects of the various variables on MACE. A P value (two-tailed) of less than 0.05 was identified as statistical significance. The variables with unadjusted $P < 0.1$ were accepted to be confounding factors and included in the backward multivariate Cox logistic regression analysis to determine the independent predictors of MACE. The predictive value of LAVI/LVEFr was estimated by the areas under the receiver operating characteristic (ROC) curve. Kaplan–Meier curve was drawn to show the LAVI, LVEF, and LAVI/LVEFr percentiles in predicting MACE.

Results

A total of 176 patients were included in the study. The mean age of participants was 61.1 ± 12.1 and 26 (14.8%) of them were female. The patients were divided into two groups according to the presence of MACE which was occurred in 70 patients (39.7%). The MACE (+) group was older (66.5 ± 11.5 vs. 57.5 ± 11.1, $P < 0.001$) and more likely to be male (77.1% vs. 21.9%, $P = 0.014$). While MACE (+) group more likely to have DM (34%. 3 vs. 18.9%, $P = 0.017$) and higher smoking rate (40% vs. 28.3%, $P = 0.034$); HL (62.3% vs. 42.9%, $P = 0.009$) rate was higher in MACE (−) group [Table 1]. In addition, MACE (+) group had higher previous PCI (20% vs. 5.7%, $P = 0.004$) and anterior MI (62.9% vs. 43.4%, $P = 0.011$) rates. Higher TIMI flow following PCI was associated with lower MACE occurrence ($P < 0.001$).

Laboratory analysis revealed that while serum creatinine level (1.02 ± 0.37 vs. 0.91 ± 0.2 mg/dL, $P = 0.012$), neutrophil count (8.4 ± 3.3 vs. 7.19 ± 3.2 10^3/μL, $P = 0.016$), peak creatine kinase MB (215 [120–300] vs. 300 [230–300] ng/uL, $P = 0.011$), and glucose level (94 [82–105] vs. 108 [82–120] mg/dL, $P = 0.008$) were higher; hemoglobin level (14.2 ± 1.5 vs. 13.6 ± 1.3 mg/dL, $P = 0.005$) was lower in patients with MACE.

Among echocardiographic findings, while LVEF was lower (38.1 ± 7.9 vs. 45.6% ±9.6%, $P < 0.001$); LAVI [26.9 [17.3–30.8] vs. 38.9 [23.3–44] ml/m^2, $P < 0.001$) and LAVI/LVEFr (97.2 ± 30 vs. 57.2 ± 25, $P < 0.001$) were higher in patients with MACE [Table 1].

Backward multivariable regression analysis was conducted with MACE relevant parameters and found that age (odds ratio [OR] =1.062, 95% confidence interval [CI]: 1.021–1.104, $P = 0.003$), serum creatinine level (OR = 6.419, 95% CI: 2.278–18.091, $P < 0.001$), and LAVI/LVEFr (OR = 1.032, 95% CI: 1.019–1.045, $P < 0.001$) were independent predictors of MACE during long-term follow-up [Table 2]. Since LAVI/LVEFr includes LAVI and LVEF, we did not combine these parameters in the regression model to avoid interaction between them in multivariable analysis. Prognostic values of these parameters were compared with area under curve (AUC) models in ROC curve analysis.

We performed ROC curve analysis and found that the AUC of LAVI, LAVI/LVEFr, and LVEF was found as 0.769, 0.874, and 0.746, respectively [Figure 1]. Kaplan–Meier curves demonstrated that higher LAVI/LVEFr increased MACE, launching from the early stage [Figure 2].

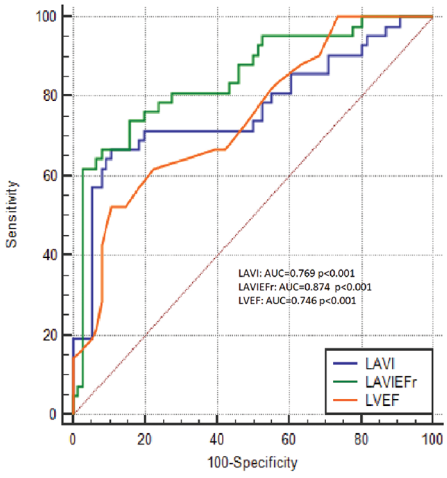

Figure 1: Sensitivity and specificity of left atrial volume index, left ventricle ejection fraction, and left atrial volume index left ventricle ejection fraction ratio

Table 1: Baseline characteristics of the study population

Variable	MACE (−) (*n*=106), *n* (%)	MACE (+) (*n*=70), *n* (%)	All patients (*n*=176), *n* (%)	*P*
Demographic data				
Age (years)	57.5±11.1	66.5±11.5	61.1±12.1	<0.001
Gander (male)	96 (90.6)	54 (77.1)	150 (85.2)	0.014
DM	20 (18.9)	24 (34.3)	44 (25)	0.017
HT	34 (32.1)	26 (37.1)	60 (34.1)	0.297
HL	66 (62.3)	30 (42.9)	96 (54.5)	0.009
Current smoking	30 (28.3)	28 (40)	56 (32.9)	0.034
Family CAD history	12 (11.3)	4 (5.7)	16 (9.1)	0.159
Previous PCI	6 (5.7)	14 (20)	20 (11.4)	0.004
BMI (kg/m^2)	27.8 (25.4-30.8)	28 (24.7-30.7)	27.8 (25.2-30.8)	0.188
PBT (s)	270 (150-450)	330 (210-390)	270 (157-390)	0.358
GRACE score	101.9±27.1	126.4±26	111±29	<0.001
Angiographic data				
Killip class				
1	88 (83)	54 (77.1)	142 (80.7)	0.219
>1	18 (17)	16 (22.9)	34 (19.3)	
Type of MI				
Anterior	46 (43.4)	44 (62.9)	90 (51.1)	0.011
Nonanterior	60 (56.6)	26 (37.1)	86 (48.9)	
IRA				
LAD	48 (45.3)	44 (62.9)	92 (52.3)	0.018
RCA	42 (39.6)	14 (20)	56 (31.8)	
CX	16 (15.1)	12 (17.1)	28 (15.9)	
Final TIMI flow				
1	8 (7.5)	2 (2.9)	10 (5.7)	<0.001
2	10 (9.4)	32 (45.7)	42 (23.9)	
3	88 (83)	36 (51.4)	124 (70.5)	
Laboratory and echocardiographic data				
Creatinine (mg/dL)	0.91±0.2	1.02±0.37	0.95±0.28	0.012
WBC (10^3/μL)	7.19±3.2	8.4±3.3	7.6±3.3	0.016
Hemoglobin (mg/dL)	14.2±1.5	13.6±1.3	14±1.4	0.005
Peak CK-MB (ng/uL)	215 (120-300)	300 (230-300)	241 (127-300)	0.011
Peak troponin (ng/uL)	44.3 (41.4-47.3)	48.7 (47-50)	45 (43-47)	0.048
Fasting glucose (mg/dL)	94 (82-105)	108 (82-120)	109 (95-159)	0.008
HsCrp (mg/dL)	0.43 (0.31-0.96)	0.95 (0.27-1.48)	0.48 (0.32-1.31)	0.070
LVEF (%)	45.6±9.6	38.1±7.9	42.6±9.7	<0.001
LAVI (ml/m^2)	26.9 (17.3-30.8)	38.9 (23.3-44)	29.6 (19.1-36.9)	<0.001
LAVI/LVEFr	57.2±25	97.2±30	71.5±33	<0.001
Medication at discharge				
ASA	104 (98.1)	68 (97.1)	172 (97.7)	0.873
Clopidogrel	102 (96.2)	67 (95.7)	169 (96)	0.772
BB	95 (89.6)	52 (89.7)	147 (89.6)	0.687
ACEI/ARB	88 (83)	57 (81.4)	145 (82.3)	0.298
Statin	102 (96.2)	68 (97.1)	170 (96.5)	0.887

DM=Diabetes mellitus, HT=Hypertension, HL=Hyperlipidemia, CAD=Coronary artery disease, PCI=Percutaneous coronary intervention, BMI=Body mass index, PBT=Paint o balloon time, MI=Myocardial infarction, IRA=Infarct-related artery, LAD=Left anterior descending artery, RCA=Right coronary artery, CX=Circumflex artery, WBC=White blood count, CK-MB=Creatine kinase-MB, HsCrp=High sensitive C-reactive protein, LVEF=Left ventricle ejection fraction, LAVI=Left atrial volume index, LAVIL/VEFr=Left atrial volume index to left/ventricle ejection fraction ratio, ASA=Acetyl salicylic acid, BB=Beta blocker, ACEI=Angiotensin-converting enzyme inhibitor, ARB=Angiotensin receptor blockers, TIMI=Thrombolysis in myocardial infarction, GRACE=Global registry of acute coronary events

Discussion

It was revealed that increased age, LAVI/LVEFr, and serum creatinine level predicted long-term MACE during 8 years of follow-up. To the best of our knowledge, this novel index, LAVI/LVEFr, was evaluated in patients with STEMI in the present study firstly.

The interest in LA has increased in recent years due to its' multifaceted property. The LA plays a pivotal role in the

Table 2: Cox regression analysis for predicting major adverse cardiovascular events

Variable	Univariate			Multivariable		
	OR	95% CI	*P*	OR	95% CI	*P*
Age (years)	1.072	1.041-1.104	<0.001	1.062	1.021-1.104	0.003
Gander (male)	2.844	1.207-6.705	0.017	1.075	0.308-3.755	0.910
DM	2.243	1.122-4.486	0.022	1.609	0.769-3.367	0.206
Current smoking	2.115	1.123-4.846	0.056	1.701	0.453-6.388	0.431
Anterior MI	2.207	1.189-4.097	0.012	1.563	0.730-3.347	0.251
Serum creatinine	4.082	1.274-13.082	0.018	6.419	2.278-18.091	<0.001
Killip score	1.449	0.682-3.078	0.335	0.666	0.269-1.651	0.380
LAVI/LVEFr	1.029	1.021-1.038	<0.001	1.032	1.019-1.045	<0.001

MI=Myocardial infarction, LAVI/LVEFr=Left atrial volume index/left ventricle ejection fraction ratio, CI=Confidence interval, OR=Odds ratio, DM=Diabetes mellitus

cardiac cycle as a functioning conduit for pulmonary venous blood during early ventricular diastole and as a booster pump, augmenting ventricular filling during late ventricular diastole. With this crucial function, LA contributes to LVEF approximately 30%.[11] In addition, LA modulates the hemodynamic balance by secreting atrial natriuretic peptide (ANP) response to stretch of the atrial wall. The ANP modulates the counterbalance between the renin-angiotensin-aldosterone and parasympathetic systems by providing natriuresis and vasodilatation. Thus, LA is also considered to be the component of the neuroendocrine system.[12] Pressure and/or volume overload in LA endorses LA enlargement. Therefore, LA indicators are also indirect indices of LV chamber compliance and diastolic function as well as of the intracardiac pressure and volume overload.[13] Although impaired diastolic function was shown to be associated with MACE in patients with MI, it was reported not to be clinically apparent in most of the cases. For that purpose, LA measurements were used to be diastolic dysfunction markers in several studies.[14,15]

LAVI has been proven to be the most reliable marker among LA enlargement indices. Elevated LAVI was shown to be associated with the worse outcome beyond the diastolic dysfunction in acute coronary syndrome (ACS), cardiomyopathies, and valvular diseases.[4] Moller *et al*. showed that elevated LAVI was related to increased mortality in patients with AMI during 2 years of follow-up.[16] Similarly, Beinart *et al*. reported that elevated LAVI was associated with mortality after 5 years of follow-up.[17] Besides, the LA enlargement promotes blood stasis and increases the risk for atrial fibrillation (AF) and stroke, consequently. AF is also a well-established risk factor for HF, stroke, and mortality particularly in patients with ACS.[18] We think that increased LAVI might be responsible for a higher MACE rate by also inducing AF. On the other hand, LA enlargement is a chronic adaptive process, and deteriorated LA function frequently accompanies LA enlargement. Therefore, in case of the acute cardiovascular clinics such as STEMI, it can be speculated that increased LAVI implies a higher atherosclerotic burden before the STEMI occurrence. However, there is not constant LAVI value evaluated in previous studies. While in one study,

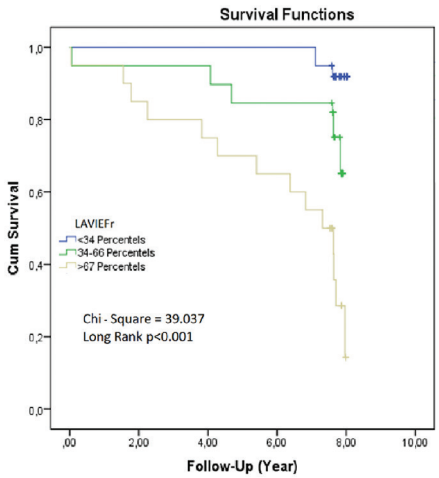

Figure 2: Kaplan–Meier curves demonstrated that higher left atrial volume index ventricle ejection fraction ratio increased major adverse cardiovascular event beginning from the early follow-up

under the "32 ml/m²"of LAVI was reported to be normal; another study considered "28 ml/m²" as median LAVI value.[5,16] Moreover, LAVI was dichotomized in many previous studies as elevated or normal. With this context, it is still needed to be validated the value of LAVI in predicting future adverse events with further trials.

The LVEF is one of the most examined echocardiographic parameters at admission and during follow-up and was found to be associated with long-term outcomes and mortality in ACS patients in previous studies.[19] Bosch *et al*. found that LVEF has more predictive value than other echocardiographic parameters predicting MACE in patients with ACS. In addition, in patients whose LVEF was under 48%, the mortality rate was 3.3-fold higher.[20] However, in case of elevated LAVI and normal LVEF or normal LAVI and decreased LVEF, we may need a further tool for better outcome prediction. In addition, in the early stage of STEMI, tachycardia, transient ischemic dysfunction, stunning, or hibernation may occur and thus, LVEF may show variability. Therefore, quantitative and combined indices use could provide a more objective and accurate outcome prediction. Indeed, it was shown that adding

LVEF to other scoring tools elicits more accurate results.[21] Besides, decreasing LA function has a minimal effect at normal ejection fraction, whereas it exacerbates HF symptoms in patients with reduced EF.[4] Therefore, given the prognostic roles of the LAVI and LVEF, we think the combined usage of them might facilitate MACE prediction as compared to their single usage.

On the other hand, HF with preserved ejection fraction (HFpEF) accounts for almost half of all types of HF. HFpEF has a high risk for morbidity and mortality, and quality of life may be worse than with HF with reduced EF (HFrEF). There is not a single objective marker to define HFpEF, and so its' diagnosis is challenging. Therefore, these patients are evaluated by measuring diastolic parameters. HFpEF is suspected in patients with symptoms and findings of HF accompanying structural heart diseases such as LA enlargement or left ventricular hypertrophy. However, it is a multisystemic disease and might present with noncardiac complaints. Therefore, patients without specific signs and symptoms of HF can easily be disregarded.[22,23] On the other hand, ACS was shown to be the risk factor for HFpEF. However, HFpEF may manifest as mild diastolic dysfunction at the early stage of ACS, but it progresses depending on the severity of the MI. Antonelli *et al*. showed that those who were developed HFpEF had a 3-fold higher mortality risk than those without HF among patients with acute MI.[24] Therefore, the parameters that imply these patients would provide more accurate evidence about surveillance for sure. In the present study, LAVI/LVEFr was a significant predictor of MACE in the fully adjusted model. We consider that LAVI/LVEFr might be indicating HFpEF and associated with increased MACE consequently. That being the case, it can be speculated that we have revealed the underlying HFpEF patients by studying the LAVI/LVEFr. Thus, this ratio can be evaluated with further studies as a novel marker in patients who were suspected of HFpEF.

The association between renal function and cardiovascular diseases is well validated. Especially, renal dysfunction is one of the most common comorbidities in patients with HF and ACS and is related to short- and long-term adverse outcomes.[25] Similar to previous studies, we showed that the creatinine predicted the MACE in patients with ACS strongly and independently. Renal function assessment may also be added to the prediction models of MACE.

Limitations

There are multiple limitations to acknowledge. It is a single-center study with a relatively limited number of patients. LAVI/LVEFr had relatively weak prognostic value.

Conclusion

This simple, easily applicable, reliable, and novel index, LAVI/LVEFr, predicted long-term MACE in patients with STEMI who underwent p-PCI. The combination of systolic and diastolic parameters may be more logical for prognosticate future adverse events due to both are associated with MACE. Besides, this ratio might be a marker of HFpEF in patients with STEMI or in the normal population.

Ethical clearance

Recep Tayyip Erdogan University Ethic Committee approved the study with 40465587-0.50.01.04-162 number and 13/07/2021 date.

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Nil.

Conflicts of interest

There are no conflicts of interest.

References

1. Collet JP, Thiele H, Barbato E, Barthélémy O, Bauersachs J, Bhatt DL, *et al.* ESC Scientific Document Group. 2020 ESC Guidelines for the management of acute coronary syndromes in patients presenting without persistent ST-segment elevation. Eur Heart J 2021;42:1289-1367.
2. Lev EI, Kornowski R, Vaknin-Assa H, Porter A, Teplitsky I, Ben-Dor I, *et al.* Comparison of the predictive value of four different risk scores for outcomes of patients with ST-elevation acute myocardial infarction undergoing primary percutaneous coronary intervention. Am J Cardiol 2008;102:6-11.
3. Klaeboe LG, Edvardsen T. Echocardiographic assessment of left ventricular systolic function. J Echocardiogr 2019;17:10-6.
4. Çetin M, Özer S, Çinier G, Yılmaz AS, Erdoğan T, Şatıroğlu Ö. Left atrial volume index and pulmonary arterial pressure predicted MACE among patients with STEMI during 8-year follow-up: experience from a tertiary center. Herz 2021;46:367-74.
5. Gunasekaran R, Maskon O, Hassan HH, Safian N, Sakthiswary R. Left atrial volume index is an independent predictor of major adverse cardiovascular events in acute coronary syndrome. Can J Cardiol 2012;28:561-6.
6. Cirakoglu OF, Aslan AO, Yilmaz AS, Şahin S, Akyüz AR. Association between C-reactive protein to albumin ratio and left ventricular thrombus formation following acute anterior myocardial infarction. Angiology 2020;71:804-11.
7. Erdoğan T, Çetin M, Çinier G, Özer S, Yõlmaz AS, Karakişi O, *et al.* Preoperative blood urea nitrogen-to-left ventricular ejection fraction ratio is an independent predictor of long-term major adverse cardiac events in patients undergoing coronary artery bypass grafting surgery. J Saudi Heart Assoc 2020;32:79-85.
8. De Boer IH, Bangalore S, Benetos A, Davis AM, Michos ED, Muntner P, *et al.* Diabetes and hypertension: A position statement by the American diabetes association. Diabetes Care 2017;40:1273-84.
9. Ahluwalia N, Dwyer J, Terry A, Moshfegh A, Johnson C. Update on NHANES dietary data: Focus on collection, release, analytical considerations, and uses to inform public policy. Adv Nutr 2016;7:121-34.
10. Levine GN, Bates ER, Bittl JA, Brindis RG, Fihn SD, Fleisher LA, *et al.* 2016 ACC/AHA guideline focused update on duration of dual antiplatelet therapy in patients with coronary artery disease: A report of the American College of Cardiology/American Heart Association task force on clinical practice guidelines. J Am Coll Cardiol 2016;68:1082-115.
11. Blume GG, Mcleod CJ, Barnes ME, Seward JB, Pellikka PA, Bastiansen PM, *et al.* Left atrial function: Physiology, assessment, and clinical implications. Eur J Echocardiogr 2011;12:421-30.
12. Lindberg S, Jensen JS, Hoffmann S, Pedersen SH, Iversen AZ, Galatius S, *et al.* Interplay between adiponectin and pro-atrial natriuretic peptide and prognosis in patients with ST-segment elevation myocardial infarction. Am J Cardiol 2015;116:1340-5.
13. Hoit BD. Left atrial size and function role in prognosis. J Am Coll Cardiol 2014;63:493-505.
14. Kobayashi A, Misumida N, Fox JT, Kanei Y. Prognostic value of left ventricular end-diastolic pressure in patients with non-ST-segment elevation myocardial infarction. Cardiol Res 2015;6:301-5.

15. El Aouar LM, Meyerfreud D, Magalhães P, Rodrigues SL, Baldo MP, Brasil Y, *et al.* Relationship between left atrial volume and diastolic dysfunction in 500 Brazilian patients. Arq Bras Cardiol 2013;101:52-8.
16. Moller JE, Hillis GS, Oh JK, Seward JB, Reeder GS, Wright RS, *et al.* Left atrial volume: A powerful predictor of survival after acute myocardial infarction. Circulation 2003;107:2207-12.
17. Beinart R, Boyko V, Schwammenthal E, Kuperstein R, Sagie A, Hod H, *et al.* Long-term prognostic significance of left atrial volume in acute myocardial infarction. J Am Coll Cardiol 2004;44:327-34.
18. Modin D, Olsen FJ, Pedersen S, Jensen JS, Biering-Sørensen T. Measures of left atrial function predict incident atrial fibrillation in STEMI patients treated with primary percutaneous coronary intervention. Int J Cardiol 2018;263:1-6.
19. Siontis GC, Branca M, Serruys P, Silber S, Räber L, Pilgrim T, *et al.* Impact of left ventricular function on clinical outcomes among patients with coronary artery disease. Eur J Prev Cardiol 2019;26:1273-84.
20. Bosch X, Théroux P. Left ventricular ejection fraction to predict early mortality in patients with non-ST-segment elevation acute coronary syndromes. Am Heart J 2005;150:215-20.
21. Perelshtein Brezinov O, Klempfner R, Zekry SB, Goldenberg I, Kuperstein R. Prognostic value of ejection fraction in patients admitted with acute coronary syndrome: A real world study. Medicine (Baltimore) 2017;96:e6226.
22. Redfield MM. Heart failure with preserved ejection fraction. N Engl J Med 2016;375:1868-77.
23. Pfeffer MA, Shah AM, Borlaug BA. Heart failure with preserved ejection fraction in perspective. Circ Res 2019;124:1598-617.
24. Antonelli L, Katz M, Bacal F, Makdisse MR, Correa AG, Pereira C. *et al*. Heart failure with preserved left ventricular ejection fraction in patients with acute myocardial infarction. Arq Bras Cardiol 2015;105:145-50.
25. Wright RS, Reeder GS, Herzog CA, Albright RC, Williams BA, Dvorak DL, *et al.* Acute myocardial infarction and renal dysfunction: A high-risk combination. Ann Intern Med 2002;137:563-70.